Air Embolism Associated with Pulmonary Artery Catheter Introducer Kit

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Catheter introducer kits provided with self-sealing or valved pulmonary artery catheter introducer ports have become commercially available to reduce the risk of air entry into the circulation during insertion of the pulmonary artery catheter and after its removal. There are also catheter introducer kits which have ports that are not self-sealing. The use of a catheter introducer kit which does not provide the self-sealing introducer port resulted in two potentially fatal episodes of air embolism in patients during the postoperative period.

REPORTS OF TWO CASES

Patient 1. A 53-year-old man was admitted for hemiglasseotomy for recurrence of a squamous cell carcinoma of the tongue and floor of the mouth. His medical history included myocardial infarctions two and three years preoperatively, a long history of angina pectoris, hypertension, chronic alcohol abuse, and an 80 pack-year history of cigarette smoking. In the operating room, a radial arterial catheter and a right internal jugular catheter introducer (Arrow International, 7-French Catheter/Sheath Adapter with Side Port, #AK-06800) were inserted percutaneously. A pulmonary artery catheter was inserted through the catheter introducer and the induction of anesthesia proceeded. Surgery was uneventful with pulmonary arterial, central venous, and arterial pressures remaining in the normal ranges throughout the course of the procedure. The trachea was extubated in the Surgical Intensive Care Unit. After several hours of observation, the pulmonary artery catheter was removed from the catheter introducer by a physician who had not been present during the insertion of the catheter introducer. The indwelling introducer site was redressed and the side port utilized for intravenous fluid administration. The patient was subsequently changed to the head-elevated position by one of the Intensive Care Unit staff.

A few minutes after the patient resumed the head-elevated position, he was noted to be tachypneic, tachycardic, and appeared dusky. Significant ECG changes were noted on the monitor in the form of ST-segment depression and premature ventricular beats. Blood pressure decreased from 130/75 to 100/60 mmHg within 1 min. Auscultation of the heart revealed a "mill-wheel" murmur characteristic of venous air embolism. While the patient was being placed in the left lateral decubitus position, the catheter introducer was removed since it was the likely source of air entry into the circulation but the exact location of the leak in the introducer was not immediately apparent. There was a transient episode of bradycardia which apparently responded to the change in position. While breathing room air, the pH was 7.38, PaCO₂ 45 mmHg, and PaO₂ 37 mmHg. With an approximate FIO₂ of 0.5 to 0.6 delivered by mask, pH was 7.34, PaCO₂ 43 mmHg, and PaO₂ 90 mmHg 10 min later. The patient recovered with no lasting complications.

Patient 2. A 64-year-old man was admitted for left profundoplasty for severe claudication secondary to thrombosis of a previous aorto-femoral bypass. His medical history was remarkable for myocardial infarction eight years preoperatively, angina pectoris, hypertension, and diabetes mellitus. After being transported to the operating room, a radial arterial catheter and a right internal jugular catheter introducer (Arrow International, 7-French Catheter/Sheath Adapter with Side Port, #AK-06800) were inserted percutaneously. The introducer was placed to permit the insertion of a pulmonary artery catheter intraoperatively since the change to a more extensive surgical procedure was likely. A 16-gauge, tapered, 30-cm central venous catheter which seals in the pulmonary artery port of the introducer (Argyle, Intramedic) was inserted through the catheter introducer and the induction of anesthesia proceeded. The operative procedure was uneventful, the trachea was extubated and the patient was taken to the Intensive Care Unit for observation.

After several hours in the Intensive Care Unit, the patient pulled the central venous catheter out of the catheter introducer. The patient was noted by the nurse to be disoriented and tachypneic. The only ECG change noted was an increase in heart rate from 95 to 125 beats/min. Blood pressure was unchanged. The patient was placed in the left lateral decubitus position and several ml of air were recovered from a central venous catheter passed through the introducer. Auscultation of the heart did not reveal a "mill-wheel" murmur. With an FIO₂ of 0.4, the pH was 7.42, PaCO₂ 38 mmHg, and the PaO₂ 56 mmHg. Six hours later the pH was 7.43, PaCO₂ 39 mmHg, and the PaO₂ 118 mmHg. The patient had no further difficulty related to the air embolus.

DISCUSSION

Venous air embolism is a well-recognized complication of invasive monitoring of the right side of the heart. In these cases all the standard precautions were taken to prevent air embolism but a peculiarity of one of the several catheter introducer kits used in our institution resulted in nearly fatal accidents in patients who had undergone successful anesthetic and surgical procedures.
The complication in the first case occurred as a result of a common practice of removing the pulmonary arterial catheter from the catheter introducer and using the side arm of the introducer as a central venous access port for drug administration. The catheter introducer kit used in these two cases has no practical provision for self-sealing of the pulmonary arterial catheter introducer site after the removal of the pulmonary arterial catheter and is easily confused with other types of introducers which have self-sealing valves. A 7-French plug is included in the sterile package which is intended for use as an obturator to prevent air entry if a pulmonary arterial catheter is not inserted into the port. The risk of air entry is clearly stated in the package insert. However, if a catheter is inserted, the plug is discarded since no provision is made for maintaining it sterile. Once the pulmonary arterial catheter is removed (frequently by personnel in the Intensive Care Unit not involved with the choice of the introducer and who have not seen the warning on the package insert) a hole at least 7-French in diameter remains (fig. 1). In the first case, this fact was not known by the physician who removed the pulmonary artery catheter and replaced the dressing. This physician confused the introducer with a self-sealing type frequently used in our hospital (Cordis®). By allowing the patient to sit after the removal of the catheter, air entry occurred through the hole resulting in a serious post-operative complication.

The risk of air entry and the safe use of the Arrow® introducer were discussed with the housestaff and nurses. Several days later, despite increased awareness of the risk, an air embolism occurred in the second patient. This time, however, the patient himself removed the catheter from the introducer port.

Large volumes of air may enter the circulation if the port is left open. Conahan1 has measured the relationship between pressure gradient and air flow through the 7-French introducer and clearly demonstrated that a 4 mmHg gradient is sufficient to produce a fatal air embolism in an adult. With a smaller gradient and slow infusion Adorno et al.2 has demonstrated that a gaseous result is evoked resulting in an increase in the pressure gradient across the catheter which may result in a bolus of air entry into the circulation, air lock, and death. English et al.3 have shown that the changes in the ECG, arterial blood gases, blood pressure, the presence of the "mill-wheel" murmur, and arrhythmia are all late intermediate or late changes seen only with 1 to 2 ml/kg of air entry into the heart suggesting that our first patient probably entrained 75 to 150 ml of air through the introducer. The large intrapulmonary shunt, hypoxia, hypotension, and ECG changes observed in the first case also suggest a significant volume of air entry. In the second patient, the increase in intrapulmonary shunt which resolved after several hours and air recovery from the central catheter suggests that a significant air leak was present. The treatment of this complication is well-described by Alvaran et al.4

Because of these unfortunate experiences with this particular catheter introducer, we encourage the use of only self-sealing catheter introducers for patients who are to be attended by several different medical teams and have warned the medical and nursing staff in other Intensive Care areas of the potential danger and easy confusion of the introducers which are self-sealing with those that require obturators or manual closure of the port. The problem with the Arrow® #AK-06800 kit may be short-lived since the manufacturer is developing a self-sealing introducer valve.

There are, however, several other types of catheter introducers available with valves which fall into three general categories: 1) not self-sealing, 2) self-sealing, and 3) adjustable orifice, not self-sealing (Tuohy-Borst®). Many are available with and without side arm ports. The Arrow® #AK-06800, Stancos®, Argon® PAC TRAY/2, Argon® PAC KIT/2, and all introducers without side arm ports (UMI®, for example) are not self-sealing. The Cordis® #501-638 Kit, all USCI® introducers with side ports, Argon® PAC TRAY/1, Argon® PAC KIT/1, and Argon® PCI KIT/4 have self-sealing valves. The Argon® PAC TRAY/3 and PAC KIT/3 contain the Tuohy-Borst® type valve mechanism which must be closed manually after the removal of the catheter.

Because supplies of catheter introducer kits which do not have self-sealing ports probably exist in many hospitals, and because the pulmonary artery catheter intro-
Second-degree Atrioventricular Block after Methyl Methacrylate

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The cardiovascular events associated with application of methyl methacrylate (MMA) consist primarily of no change or a transient decrease in blood pressure.1-3 The mechanism for this decrease is probably peripheral vasodilatation, caused by absorption of free MMA monomer into the systemic circulation.3 Cardiac arrhythmias have not been described following application of MMA, except in association with profound cardiovascular collapse.2 However, we observed a second-degree atrioventricular block in close temporal relation to MMA application during two separate operative procedures involving one patient. A detailed electrophysiologic investigation after the second occurrence failed to demonstrate intrinsic conduction system disease.

REPORT OF A CASE

A 29-year-old, 84-kg woman with rheumatoid arthritis of six years duration was scheduled for a Wagner resurfacing procedure on her left hip. Her past medical history revealed idiopathic hypothyroidism, clinically euthyroid on replacement therapy. Her physical examination, with the exception of changes related to her arthritis, was unremarkable and her preoperative laboratory examinations including ECG, chest roentgenograph, serum electrolytes, and thyroid function studies were all within normal limits. Her medications included 10 mg prednisone per day; 0.2 mg sodium levothyroxine per day; 2.4-4.8 mg enteric-coated aspirin per day in divided doses, and 500 mg naproxen twice daily. Diazepam, 15 mg, 30 cc sodium citrate orally, and 100 mg hydrocortisone succinate intramuscularly were given 90 minutes prior to induction of anesthesia. In the operating room, monitoring was established with precordial stethoscope, blood pressure cuff, and continuous electrocardiography. Thiopental, 4 mg/kg and 1 mg/kg succinylcholine were given iv, and the trachea was intubated; anesthesia was maintained with an inspired concentration of 70 per cent N₂O, 0.5 per cent enflurane, and the intravenous administration of 0.1 mg/kg diazepam and 4 μg/kg fentanyl.

Ninety minutes later, the acetalubar portion of the joint prosthesis was seated with MMA in the standard fashion. Approximately five minutes later, a 2:1 second-degree atrioventricular block (fig. 1) with a ventricular rate of 30 beats/min appeared over a 30- to 45-second period and resolved spontaneously, though 0.6 mg atropine was administered 5 to 10 seconds before the dysrhythmia resolved. The arterial blood pressure did not change and analysis of arterial blood gases and serum electrolyte concentrations were within normal limits. No similar episode occurred with the application of the femoral portion of the prosthesis. The remaining operative course proceeded uneventfully.

One month after the initial procedure, a similar operation was performed on the contralateral hip. Her anesthetic management was nearly identical with that administered during her first operative procedure. Approximately five minutes after application of MMA for the acetalubar portion of the joint prosthesis, a transient 10-second period of 2:1 atrioventricular block occurred (fig. 2) which resolved spontaneously. As before, analysis of arterial blood gases and serum electrolytes acutely were within normal limits. No conduction disturbance occurred with placement of the femoral portion of the prosthesis.

After the second occurrence of second-degree atrioventricular block, a cardiology consultation was obtained. The cardiac examination revealed no abnormality, with the exception of a grade II/VI systolic

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FIG. 1. Onset of atrioventricular block occurring during initial operative procedure.